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CASE REPORT

Postoperative developmental dural arteriovenous fistula

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Summary Dural arteriovenous fistulas (DAVFs) are rare vascular abnormalities that comprise 10—15% of all intracranial arteriovenous malformations. Strong evidence suggests that the development of DAVFs in adults is acquired and may be caused by various factors, including sinus thrombosis, venous hypertension, trauma, and infection. We report a case of a 39-year-old woman with a left temporal hemorrhagic lesion and an associated developmental venous anomaly. The patient underwent craniotomy and surgery for the removal of the mass. Approximately 2 months postsurgery, the patient complained of left-sided tinnitus with a bruit over the left temporal region. Cerebral angiography showed a DAVF close to the operation site.

1. Introduction

Dural arteriovenous fistulas (DAVFs) are acquired lesions potentially caused by various factors, including sinus thrombosis, venous hypertension, trauma, and infection. DAVFs may develop postoperatively and, in rare cases, after radiosurgery. *De novo* formation of a DAVF following supratentorial surgery is rare. We report a case of a 39-year-old woman who underwent temporal lobe surgery, after which she developed a postoperative DAVF near the operation site, and discuss the pathogenesis in light of current literature.

2. Case report

A previously healthy, 39-year-old woman presented with a severe headache in the left temporal region that had persisted for approximately 2 weeks. The associated symptoms included vomiting, stiffness in the neck, and lower back

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pain. She visited a local hospital and underwent computed tomography (CT) of the brain despite the absence of any abnormal findings on physical and neurological examinations. The CT scan revealed a lesion of 3 cm × 2.5 cm with a peripheral hyperdense zone, suggesting a hematoma of the left temporal lobe. On the following day, magnetic resonance imaging (MRI) showed a hematoma equivalent in size to that identified by the CT scan. The MRI scan also showed a T1 proton relaxation enhancement by methemoglobin on the T1-weighted image (T1WI). Furthermore, a dark signal intensity rim was apparent on the T2WI, which resulted from the T2 proton relaxation enhancement effect of hemosiderin. A suspected cavernous hemangioma adjacent to the hematoma with mixed bright and dark signal intensities on the T1WI and T2WI was noted. In addition, a concomitant developmental venous anomaly (DVA) with a tube-like enhancement of an engorged vein in front of the hematoma in the anterior temporal region was observed (Fig. 1). Cerebral angiography of the left internal carotid artery showed a mass effect on the temporal lobe with axis elevation of the middle cerebral artery. An upwardly displaced superior limb of the Sylvian triangle, and a suspected DVA at the proximal tributary of the vein of Labbé were also found; however, neither the tumor vessels nor staining were visualized (Fig. 2).

The patient refused the suggested surgery, and therefore conservative management was commenced. Approximately 1 month later, she experienced seizures twice and was transferred to the emergency department of the same hospital to which she had previously presented. A CT scan of the brain revealed a 3 cm × 2.7 cm hematoma of the left temporal lobe. She was referred to our hospital and an emergency operation was conducted. The hematoma was completely removed and histologically diagnosed as a cavernous hemangioma. The postoperative course was uneventful, and the patient was followed up at the outpatient department of our hospital.

Approximately 2 months postsurgery, the patient complained of tinnitus in her left ear with a bruit over the left temporal region. An MRI scan of the brain and cerebral angiography were conducted. The MRI scan of the brain showed a residual hematoma with a prominent vascular enhancement in the left temporal lobe, adjacent dural thickening with marked enhancement, and hypervascularity of the left temporal muscles (Fig. 3). Cerebral angiography showed a DAVF with major feeders from the left middle meningeal artery (MMA) and an ascending pterygoid branching of the left internal maxillary artery, as well as a dural branching of the left internal carotid artery and left ophthalmic artery. We found two routes of abnormal shunting and venous return, draining superiorly through the Sylvian—Trolard vein bridging into the superior sagittal sinus and another draining inferiorly through the cavernous sinuses and the inferior petrosal sinuses into both sides of the internal jugular veins (Fig. 4).

Embolization was conducted using the transvenous approach through the internal jugular veins, inferior petrosal sinuses, and cavernous sinuses to the fistula sac, and 15 detachable coils were deployed. In addition, transarterial embolization was conducted at the left MMA with 25% glue injection and at the accessory MMA with contour particles of 150—250 μm. A near-total occlusion of the arteriovenous fistula with preserved normal cortical drainage of the left cerebral hemisphere was conducted at another medical institution.

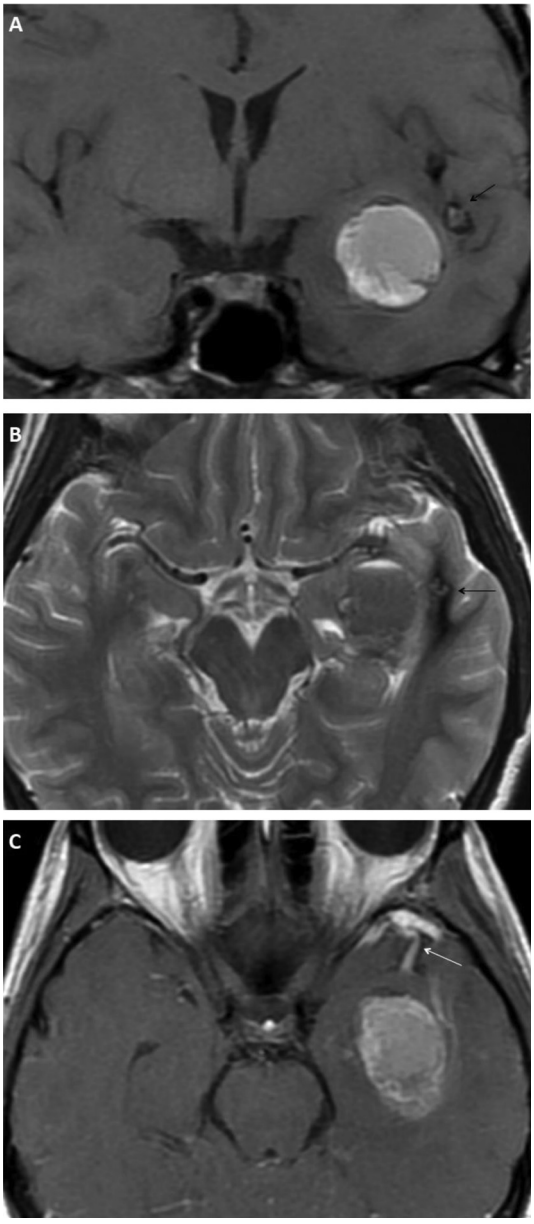

Figure 1 (A) Coronal T1-weighted image (T1WI); (B) axial T2WI revealing a hematoma of 3 cm × 2.5 cm. A suspected cavernous hemangioma adjacent to the hematoma noted with mixed bright and dark signal intensities on both T1WI and T2WI (black arrow); (C) contrast-enhanced T1WI revealing a concomitant developmental venous anomaly with a tube-like enhancement of an engorged vein in front of the hematoma in the anterior temporal region (white arrow).

3. Discussion

DAVFs are rare vascular abnormalities that comprise 10—15% of all intracranial arteriovenous malformations. Strong evidence suggests that the development of DAVFs in adults is

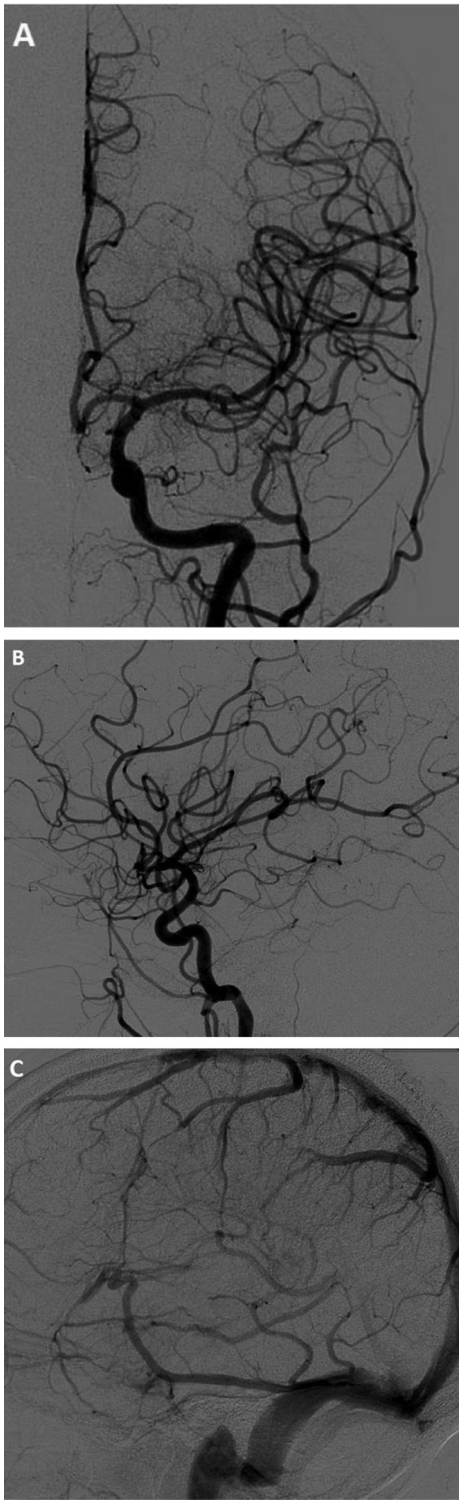

Figure 2 (A) Anteroposterior view of the left common carotid angiogram; (B) lateral view of the left common carotid angiogram; (C) lateral view of the right common carotid angiogram showing a mass effect on the temporal lobe, but no visualization of the tumor vessels or stain, (A, B). A suspected developmental venous anomaly at the proximal tributary of the vein of Labbé was also found (C).

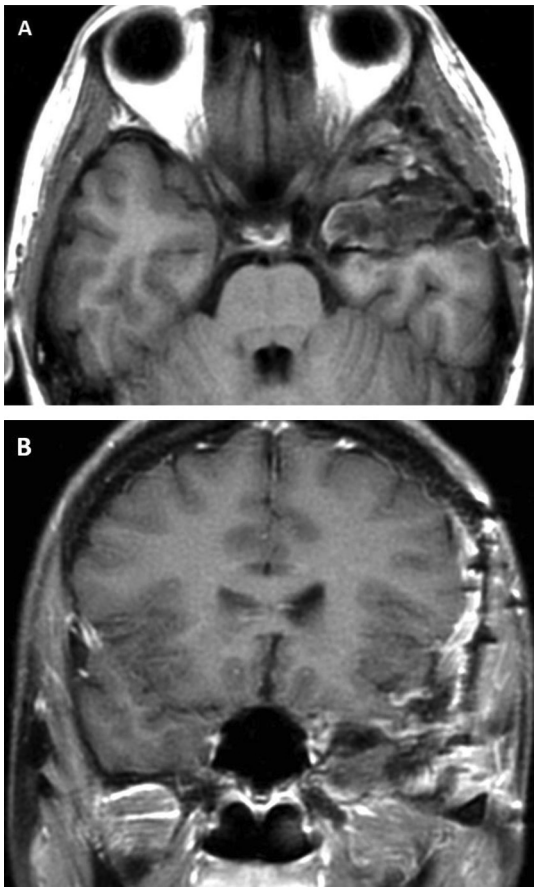

Figure 3 (A) Axial T1-weighted image showing a residual old hematoma. (B) Coronal enhanced T1-weighted image revealing a prominent vascular enhancement at the left temporal lobe, as well as adjacent dural thickening with a prominent enhancement and hypervascularity of the left temporal muscle.

acquired. Several studies have cited the presence of a normal transverse sinus on early angiograms, subsequent thrombosis of the sinus, and the later development of a DAVF proximal to the point of thrombosis.[1] Ushewokunze et al[2] reported a case of a postoperative DAVF following translabyrinthine resection of an acoustic neuroma and proposed that varying degrees of compression of the sigmoid sinus may occur during surgery. This may lead to dural venous sinus occlusion and the development of a DAVF.[2]

Shunting of arterial blood into a venous circuit raises the venous pressure. This increased pressure leads to myointimal hyperplasia of the sinus walls, narrowing of the sinus channel, and increased resistance in the sinus. Because the walls of a pia vein are weaker than those of a dural sinus, the increased pressure may lead to rupture and intracranial hemorrhage, which are generally observed clinical presentations in such cases. The venous hypertension is transmitted to the venules and capillaries, resulting in parenchymal hypoxia and vasogenic edema. Vilela et al[3] cited two cases of neoplastic occlusion of a sinus with a DAVF more proximal to the points of occlusion. These observations support the theory that the occlusive process leads to an increased venous pressure, which predisposes it to opening the existing microshunts

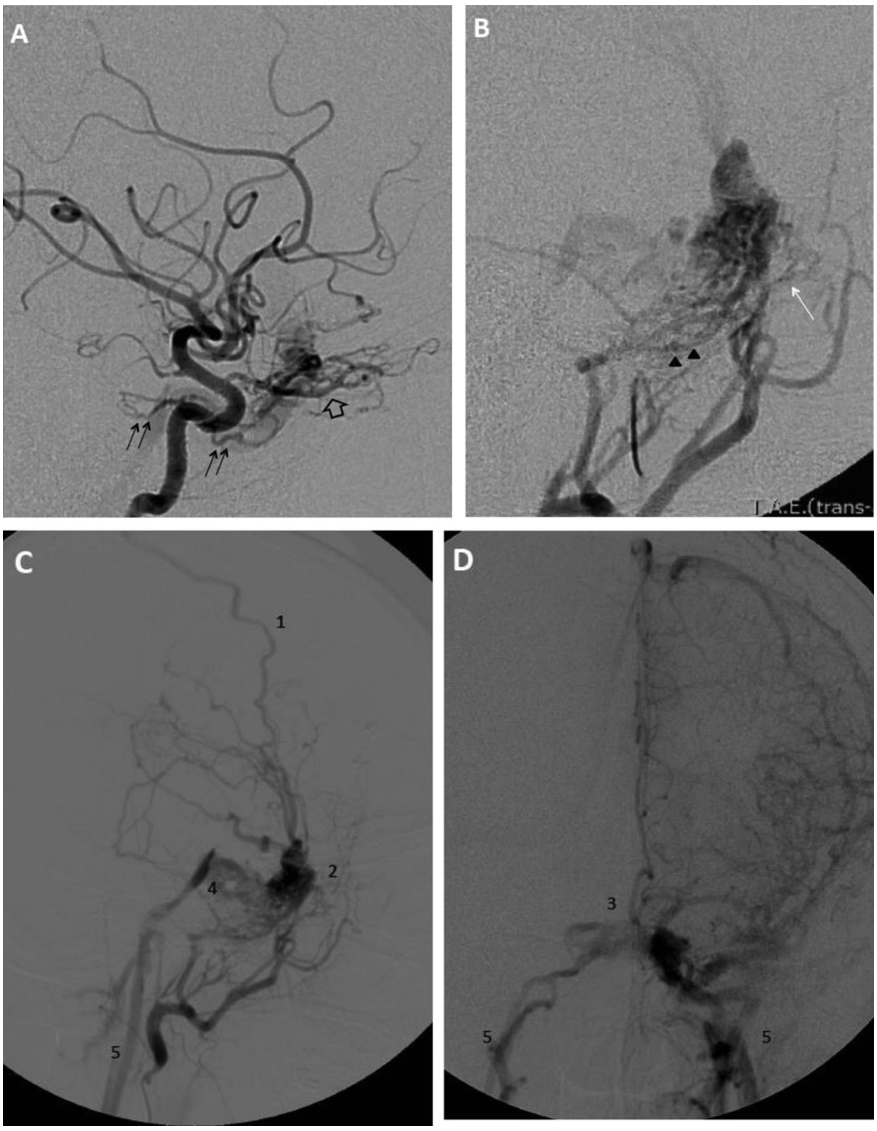

Figure 4 (A) Lateral view of the left internal carotid angiogram; (B) lateral view of the left external carotid angiogram; (C) lateral view of the left internal maxillary angiogram; and (D) anteroposterior view of the left common carotid angiogram show a dural arteriovenous fistula with major feeders of the left middle meningeal artery (arrowheads) and ascending pterygoid branching (white arrow) of the left internal maxillary artery (B) as well as the dural branching (double black arrows) of the left internal carotid artery and the left ophthalmic artery (empty arrow) (A). (C, D) Two routes of abnormal venous drainage, one draining superiorly through the Sylvian—Trolard vein (1) bridging into the superior sagittal sinus and the other draining inferiorly through the cavernous sinuses (2), circular sinus (3), and inferior petrosal sinuses (4) on both sides of internal jugular veins (5).

within the dura, and leads to the development of new shunts through angiogenesis.[1]

The present case is of a 39-year-old woman with a left temporal hemorrhagic lesion and an associated DVA. The patient underwent craniotomy and surgery for removal of the mass. Dudeck et al[4] reported a case wherein a DAVF developed in the proximity of a preexisting DVA after resection of a brainstem cavernoma. Wilson[5] hypothesized that restriction of venous outflow or undampened transmission of acute increases in venous intracranial pressure (ICP) through large radicles of a DVA results in transient or sustained elevation of pressure within this lesion. Such pressure overload transmitted to a capillary network because of a DVA during surgery might have induced the development of the DAVF. Wilson[5] also speculated that an inconspicuous thrombosis of one of the radicles of the DVA could cause an increased pressure within the DVA, promoting the *de novo* formation of a DAVF.

The postoperative development of a DAVF occurs generally at the operation site as well as at sites remote from the region of the craniotomy. These observations have led to the hypothesis that changes in the ICP or venous occlusion caused by surgery, or changes in intracranial blood flow because of tumor removal, may be the mechanisms underlying the development of a DAVF. Tissue damage and vessel injury that occur during surgery cause an inflammatory reaction and initiate a repair process with neovascular formation. Cutting the vessels may cause tissue perfusion insufficiency, venous return blockage, and disturbance to the lymphatic flow, resulting in tissue

hypoxia and edema. Chronic local hypoperfusion secondary to high intracranial sinus pressure is the major cause of angiogenesis in the dura mater, leading to the formation of an intracranial DAVF.

In conclusion, the physiopathogenesis and incidence of a postoperative developmental DAVF remains unclear. However, factors such as changes in ICP or intracranial blood flow caused by surgery and chronic local hypoperfusion, and the potential role of the DVA, may contribute to the development of a DAVF. *De novo* formation of a DAVF following supratentorial surgery is rare. The present case provides clear radiographic proof of an acquired postoperative developmental DAVF. Although *de novo* formation of a DAVF after supratentorial surgery is uncommon, surgeons should preoperatively warn their patients of this morbidity.

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